STUDY OF ELECTROCARDIOGRAPHIC CHANGES IN ACUTE ISCHEMIC STROKE

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" STUDY OF ELECTROCARDIOGRAPHIC CHANGES IN ACUTE ISCHEMIC STROKE "

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TIA	Transient ischemic attack
ECG	Electrocardiography
WHO	World health organization
CVA	Cerebrovascular accident
PCA	Posterior cerebral artery
MCA	Middle cerebral artery
ICA	Internal carotid artery
CBF	Cerebral blood flow
ICP	Intracranial pressure
CPP	Cerebral perfusion pressure
RMP	Resting membrane potential
ATP	Adenosine tri phosphate
ADP	Adenosine di phosphate
CAD	Coronary artery disease

ABSTRACT

Cerebrovascular events are a major cause of morbidity and mortality worldwide. The primary reason is atherosclerotic disease of the cerebral arteries. Other causes are embolism, haemorrhage and hypo perfusion. ECG changes simulating myocardial ischemia are common accompaniments. Objectives is to study the pattern of ECG changes in patients with Acute Ischemic Stroke. This was a cross-sectional descriptive study. The cases were taken from patients hospitalized in our hospital aged over 18 years, and 60 consecutive patients fulfilling the clinical definition of stroke were included in the study. Imaging CT/MRI confirmed the diagnosis of stroke**.**

A 12 lead electrocardiogram was recorded on admission. The study period was from January 2021 to June 2022. The results were pooled**,** and statistical analyses were performed. ECG was evaluated for different parameters like Rate, Rhythm, T wave abnormalities, QTc abnormalities, ST segment abnormalities. Mean age of presentation was 60 years. The most common ECG changes observed QTc prolongation seen in 48.3 % cases. Other ECG changes were T wave inversion in 28.3%, Bradycardia in 26.7%, ST depression in 18.4 %, and Tachycardia in 13.3% . The study confirms the role of ECG in workup of acute ischemic stroke patients. Electrocardiographic changes are very common in cases of acute ischemic stroke, even in those having no history of coronary heart disease ,understanding that these ECG which are occurring in patients with CVA is important because it may lead to erroneous judgment of assigning these patients as CAD. Interpreting these ECG changes can aid the treatment of patients with respect of revascularization and surgical interventions.

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INTRODUCTION

The 2nd most common cause of death is a STROKE. It is the fourth most common cause of disability in the world. Five million people will die from strokes yearly, affecting nearly twenty million people. Stroke-related deaths are no longer common in developed countries. Stroke deaths account for 85,5% of all fatalities in underdeveloped nations. [1] Stroke morbidity in developing nations was almost seven times higher than in industrialized nations.

According to recent studies, cerebrovascular accidents in India were the cause 0.5-9% of neurological admissions. Nine percent of stroke cases die after leaving the hospital. The fatality rate jumps to 20% at the end of one month.

A stroke is dueto reduced vascular blood flow to the brain. Cutting oxygen and glucose causes permanent damage to the brain parenchyma. A stroke is "the abrupt development of clinical signs and symptoms of a localized neurological impairment lasting more than 24 hours or resulting mortality with no clear cause other than a vascular origin," according to the WHO

They are responsible for between 80% and 85% of cerebrovascular incidents globally. [3] The constriction of blood arteries in the head or neck is one of the most frequent causes of ischemic stroke. Atherosclerosis and cholesterol accumulation are the leading causes of artery constriction. Blood stagnation happens as the vessels narrow more and more. Blood clots subsequently form as a result of this. These blood clots may dislodge and become lodged in the distal zone (embolism), obstructing the blood arteries at the

development site and inflicting ischemia damage to the brain regions.

ECG abnormalities are more common in patients with stroke. The changes in the ECG were thought to be caused by ongoing sympathetic stimulation, direct disruption to the cardiac innervation, a mismatch between the right and left sympathetic plexus, or a silent, undetected underlying heart condition. [3.] Tonic calcium channel openings, repolarization issues, and abnormal ECG patterns can all be brought on by excessive catecholamines over activating beta-adrenergic receptors..

As a result, physicians are forced to examine ECGs in patients with acute stroke since they can look like those from myocardial infarction or ischemia. They should be aware that these modifications occur in patients with acute stroke and not because of myocardial infarction4. The current study examines ECG alterations in patients with acute ischemic stroke.

AIMS AND OBJECTIVES

TO STUDY THE PATTERN OF ECG CHANGES IN ACUTE ISCHEMIC STROKE

REVIEW OF LITERATURE

In recent studies, several researchers studied ECG changes in acute ischemic stroke and its prognosis.

1. Rambabu MV et al. study showed (2018) ECG among patients with acute stroke it was found that T wave inversion was the most common ECG change found (33%), ST segment depression (10%), QRS complex changes (8%), 4
2. ECG changes observed in Gurupalsingh Sachdev (2018) study were T wave inversions(35%),QTc prolongation (23%) and ST depression (26%)1,bradycardia (8.33%),tachycardia(11.6%),atrial fibrillation (3.33%),and presence of U waves (6.66%)5
3. Ischemia, like ST changes and T wave inversions, are quite common in acute ischemic stroke. It has been reported the advancing age of presentation may itself be responsible for these changes 5
4. Toga M et al. found that the T wave abnormality (39.9%) was the most common ECG change, followed by the presence of arrhythmias (27.1%) and prolonged qt interval (32.4%), and the authors also found that T wave inversion present in 33% of the cases.6

5. Piyush Saxena's study showed (2016) ST segment and T wave changes (33%), normal ECG (28%), expired subjects with ST segments and T wave changes(47%), and normal ECG (28%). These observations suggest that these ECG changes had high mortality (poor prognosis) than acute stroke patients with normal ECG, and every patient with these changes in ECG may not have concurrent Myocardial ischemia7

6. Dogan study showed (2004), peaked, flat, inverted T wave(50%), ST segment changes (41%), the presence of these changes can be an independent predictor of early mortality8

7. Burch and colleagues[9]showed abnormal T waves, abnormal U waves, and prolong QTc in patients with stroke.

8. Crop and Manning[10],abnormal T waves were present , QTc prolongation, and ischemic ST segment .

9. Kraus , coworkers[13]noted a high abnormality of ECG in CNS lesions.

10. Miller and Abildskov revealed widespread ST-T wave irregularities. They found a lot of notched T waves.

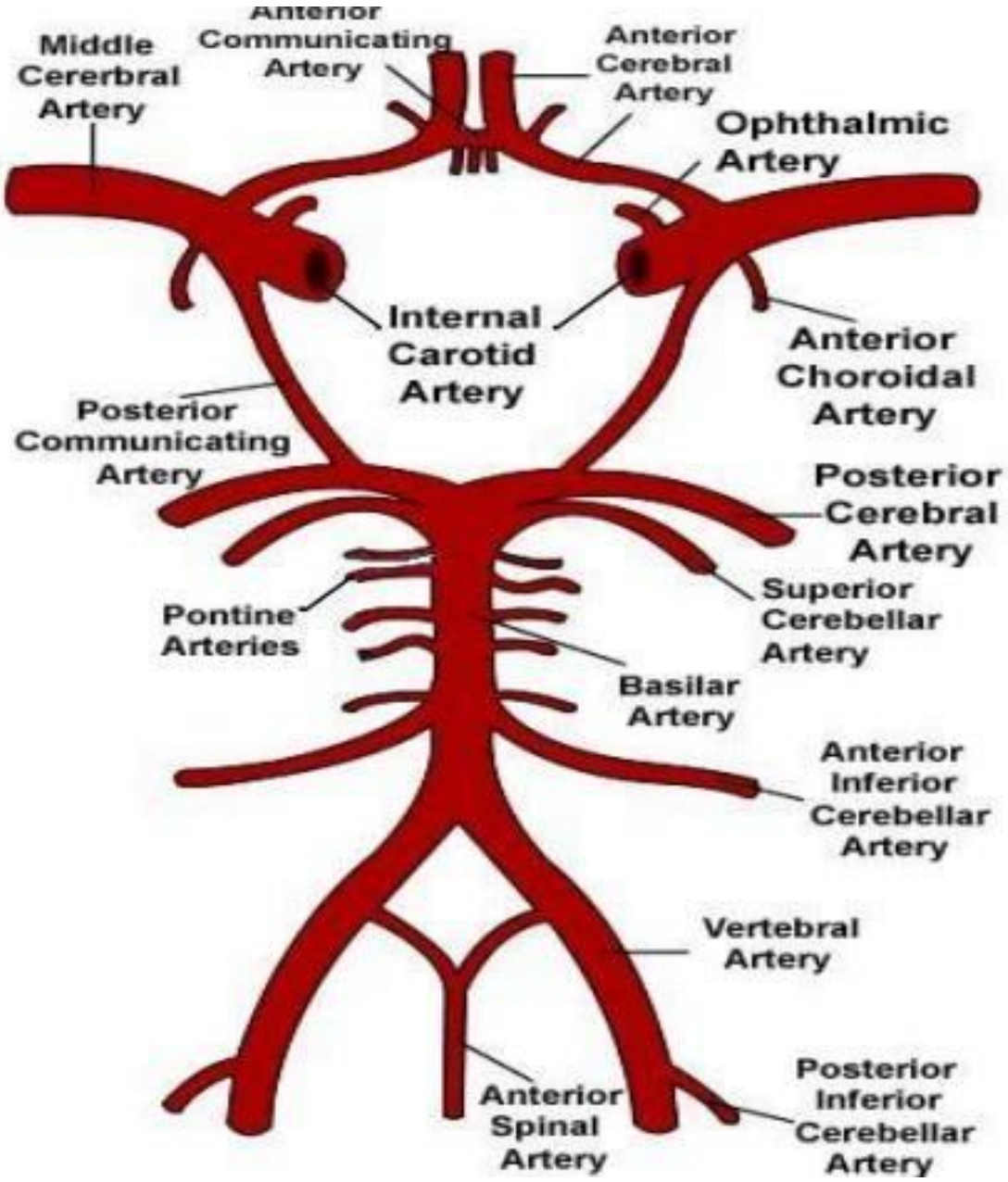
According to the WHO, a stroke is a "Rapid development of clinical signs and symptoms of a focal neurological impairment lasting longer than 24 hours or leading to death with no clear cause other than vascular origin" (Rapid development of clinical signs and symptoms). [17,18]Transient ischemic attack is used when the focused neurological impairments only last for a brief period (often less than an hour) (TIA). It ranks as the second most frequent cause of death among adults. Based on the pathophysiology of a stroke, two categories have been identified.

Thrombotic stroke: A type of stroke in which the development of thrombi causes the blood vessels in the cerebral circulation to become occluded. This kind of blood artery thrombosis accounts for around half of all stroke cases. Vascular thrombosis affects both significant and small blood vessels in cerebral circulation. In the ACA, PC, and MCA, thromboses, in equally significant areas—different areas of the brain experience lacunar infarct due to thrombosis in small penetrating arteries.

Embolic Stroke. The blood arteries in the brain carry blood clots from other bodily organs. The blood clot (emboli) typically develops from a heart source or the carotid artery and travels into the cerebral circulation. These clots become stuck inside the cerebral circulation, at the location where the artery narrows or bifurcates, and prevent blood supply to the brain parenchyma, leading to acute deficits or transient ischemic attacks, which are both types of stroke.

ARTERIAL SUPPLY OF BRAIN

Each ICA has two primary branches, the middle and anterior cerebral arteries. The basilar artery is formed by two vertebral arteries ascending and coming together at the bottom edge of the pons. The basilar artery is located near the bottom edge of the pons, midline ventral to the pons. Into two posterior cerebral arteries, it divides. The posterior communicating arteries link the vertebrobasilar and internal carotid systems. An anastomosis is connection to the base of the brain due to the anterior communicating artery connecting the two anterior cerebral arteries.



CEREBRAL BLOOD FLOW

The blood supply to the brain at any one time is known as cerebral blood flow or CBF. The average CBF in adults is 750 milliliters per minute or 15% of the cardiac output. According to this, brain tissue is exposed to 50 to 54 milliliters of blood per minute. To meet the metabolic needs of the brain, CBF is strictly controlled. blood supply to the brain reducebelow 18 - 20 ml/ 100 g/ min, it causes insufficient blood flow (ischemia), and tissue death happens if flow falls less than 8 - 10 ml / 100 g /minute.

When brain parenchyma becomes ischemic, a biochemical sequence sets in known as the ischemic cascade is initiated, which may cause brain cell death and damage. When treating patients with illnesses like shock, stroke, and traumatic brain injury, medical personnel must take precautions to preserve adequate CBF. Blood viscosity, blood vessel dilatancy, and cerebral perfusion pressure—a measure of the net pressure of blood flowing into the brain and determined by both intracranial and systemic blood pressure—are some variables that affect cerebral blood flow.

In a process known as "autoregulation," cerebral blood arteries can modify the amount of blood that flows through them by changing the diameter of their walls; they constrict when the SBP is elevated and dilate when it is decreased

CBF = CPP / CVR

PHYSIOLOGICAL BASIS OF ELECTROCARDIOGRAPHY

The electrocardiogram (ECG) is a visual representation of the electrical activity of the heart that is captured from the body surface by electrodes placed to reflect the activity from different spatial angles19.

The following elements contributed to the development of the electrocardiogram20:

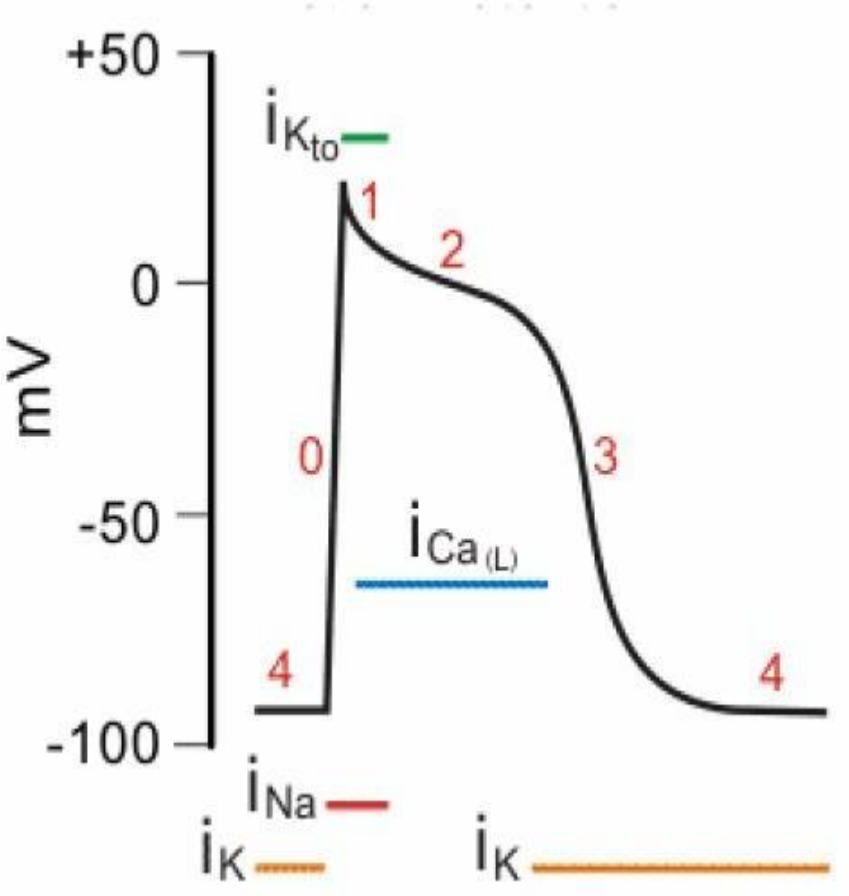
generation of impulses in the main pacemaker (sinus node).

the heart's specialised conduction mechanism for the transmission of impulses.

Depolarization and activation of the ventricular and atrial myocardium.

All of the aforementioned locations have recovered (repolarized).Intracellular potentials

The RMP of myocardial fibres is -90 mV. The gradient of K+ across the cell membrane is the main determinant of RMP. K+ is present in the body at a 30 to 45 times higher quantity intracellularly than extracellularly. For sodium (Na+), on the other hand, there is an opposing gradient. Na+ is present in greater concentrations outside of cells, ranging from 10 to 15 times more than inside cells. The permeability of the cell membrane to sodium ions (and calcium ions to a lesser extent) changes abruptly during the beginning of depolarization of the cardiac muscle cell, leading to the sudden rise in the intracellular potential to +20m V. phase 0 and stands for the first inward current11 After depolarization, the intracellular potential slowly and gradually returns to RMP (phase 4). Three phases make up repolarization in this context.



Phase 1: An initial rapid return of intracellular potential to 0 m V. This results mainly from the abrupt closing of sodium channels.

Phase 2: A plateau phase of repolarization due to entry of calcium ions into the cell.

Phase 3: This represents the slow, gradual return of intracellular potential to RMP. It results from the extrusion of potassium out of cells, establishing normal negative resting potential.

To restore the original ion concentration in the cell membrane, the sodium-potassium pump mechanism becomes effective. The energy required for this pump is derived from the

conversion of ATP to ADP. This pump removes sodium from the cell and permits potassium influx.

The sodium-potassium pump mechanism starts to act in order to return the ion concentration in the cell membrane to its initial state. The process that turns ATP into ADP supplies the power needed for this pump. This pump allows potassium to enter the cell while removing sodium from it.

ELECTROCARDIOGRAPHIC LEADS

1. Frontal plane leads: I, II, III and aVR., aVL., aVF.
2. The horizontal plane leads: V1 to V6[12].

BIPOLAR STANDARD LEADS

The bipolar standard leads I, II, and III are the original leads selected by Einthoven to record the electrical potential in the frontal plane. Einthoven placed the recording electrodes on the right arm, left arm, and left leg[21].

The bipolar leads represent a difference in electrical potential between two selected sites.

Standard lead I: This lead is derived from the placement of the negative electrode on the right arm and the positive electrode on the left arm.

Standard lead II: This lead is derived from the placement of the negative electrode on the right arm and the positive electrode on the left foot.

Standard lead III: This lead is obtained by placing a negative electrode on the left arm and the positive electrode on the left foot.

The electrocardiographic machine is provided with a right leg electrode which acts as a ground wire and plays no role in producing ECG.

AUGMENTED LEADS:

aVR, aVL and aVF are the three augmented leads that record complexes representing electrical activity at a particular point[22].

Lead aVR: Augmented unipolar right arm lead. This lead faces the heart from the right shoulder. This lead is usually oriented to the cavity of the heart. Thus all deflections – P, QRS, and T deflections are generally negative in this lead.

Lead aVL: Augmented unipolar left arm lead. This lead faces the heart from the left shoulder. It is oriented to the anterolateral or superior surface of the left ventricle.

Lead aVF: Augmented unipolar left leg lead. This lead is considered to be oriented to the inferior surface of the heart[21].

PRECORDIAL LEADS (CHEST LEADS)

The Chest leads record the electrical activity from the Chest. There are six Chest leads V1-V6 placed at different places on the Chest.

V1- fourth intercostal space at the right sternal border[23].

V2- fourth intercostal space at the left sternal border.

V3- Midway between leads V2 and V4 electrode positions.

V4- Fifth intercostal space in the left midclavicular line.

V5- Fifth intercostal space in the left anterior axillary line

V6- Fifth intercostal space in the left midaxillary line[23].

NORMAL ECG OVERVIEW[24]

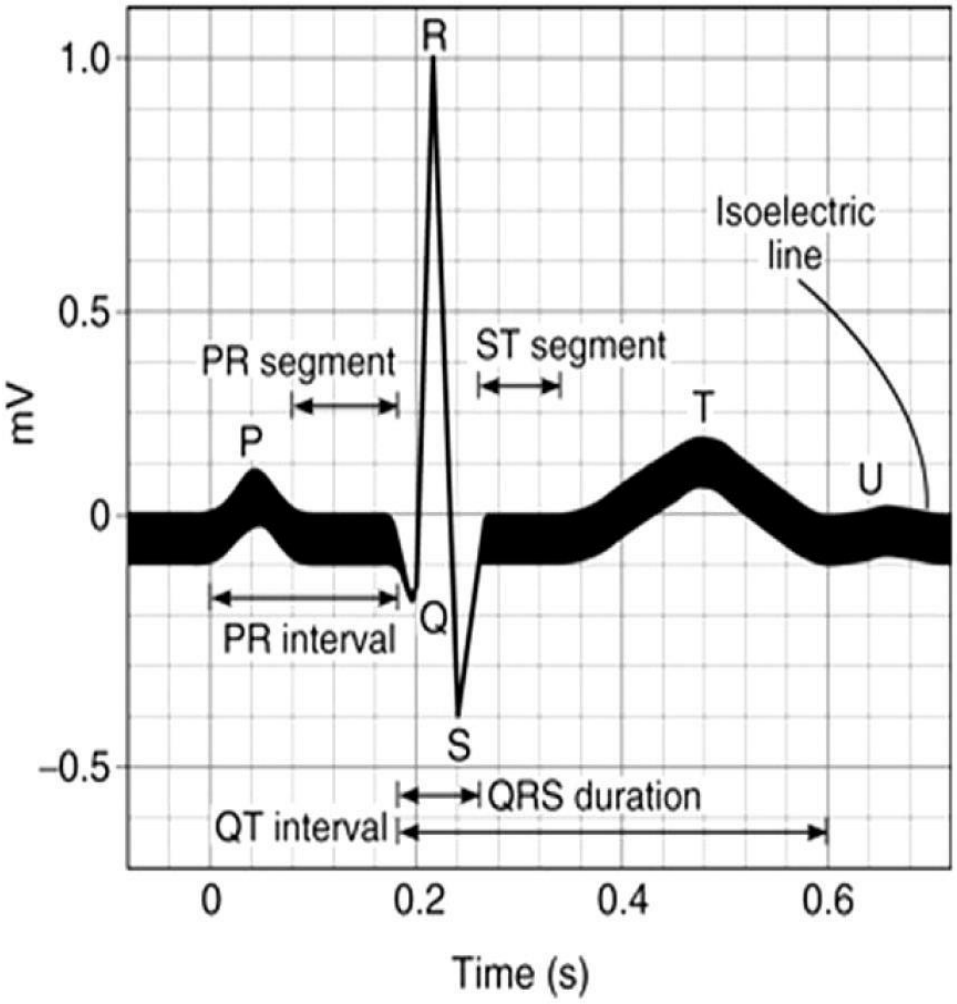


Figure no.2 Electrocardiogram.

P WAVE-

The frontal plane P wave axis in lead v1 and lead II are the best ways to evaluate a normal P wave. Lead II P wave has a pyramidal shape. P wave in the lead II lasts for 0.08 to 0.10 seconds.

Lead II has a maximum amplitude of 2.5 mm, while lead v1 has a biphasic P wave.

Duration is 0.05 seconds and is not longer than 0.08 seconds.

T WAVE

Ventricular repolarization, which typically occurs upright in left-oriented leads, causes deflection. Lead T wave v6 > v1

In lead v2-v4, there are large T waves. The persistent juvenile pattern in adults comprises inverted T waves in leads v1 to v3.

Significant T wave amplitude is defined as greater than 5 mm.

U WAVES

Normal U waves often have the same polarity as T waves and are tiny, rounded deflections of 1mm that follow T waves. Medications or hypokalemia are the most frequent causes of an aberrant rise in u wave amplitudes.

Q WAVES

Q wave is prolonged than 0.04 seconds, or that occupies at least 25% of the QRS complex, assuming that the R wave is longer than 5 mm.

ST SEGMENT

Represents a more significant portion of ventricular repolarization and departs from the baseline immediately after taking root after the QRS. When the ST segment 1mm above / below the isoelectric line in either the limb lead or the precordial lead, this is abnormal ST segment

QT INTERVAL

The duration between the start of the QRS complex and the end of the T wave represents the overall amount of ventricular activity. Ventricular depolarization and repolarization are added together in this.

METHOD OF MEASURING QT INTERVAL:

Because it could be challenging to pinpoint the precise start and finish of the interval, it might occasionally provide some difficulties. The lead I, II, AVL, and v5, v6 leads are the best indicators of the start of the QRS complex.

The notch and dip between the T and U waves are regarded as the end of the T wave when the QT interval is calculated from a lead where the U wave is dominant.

With tachycardia, the QT interval gets shorter, and with bradycardia, it gets longer. This QT lengthens when the R-R INTERVAL is reduced and shortens when the R-R INTERVAL is increased

CORRECTION OF QT INTERVAL

The QTC interval is the correct QT interval. For QT interval adjustment, numerous equations have been presented.

The bazett is most usually utilized.

QTc = QT/ root (RR) interval K constant K=QT/RR.

K usually ranges between 0.39 and 0.04 seconds. The usual range is 0.35 - 0.43 seconds.

ECG changes are present in 92 % of acute stroke individuals.[25] On the ECG of four stroke patients, Byer and colleagues first noted pronounced QT prolongation with large T and U waves in 1947. [26] Burch and colleagues later characterized an ECG pattern following a stroke that has come to be associated with cerebral vascular injury and includes T wavesabnormal, QT intervals increased[26]

QT prolongation is a myocardial abnormality that raises the risk of certain cardiac arrhythmias that can be fatal. [27]Compared to 38 percent of patients with ischemic strokes, a prolonged QT interval is frequently seen in stroke patients with ischemic [25, 28,29,30]

In individuals with ischemic stroke, QT prolongation frequently precedes ventricular tachyarrhythmia, which includes sudden death. [29, 31]

REPOLARIZATION ABNORMALITIES

The repolarization abnormalities involving the ST segment in ECG changes related to heart disease and those related to CVA are particularly striking, leading many researchers to hypothesize concomitant cardiac disease as the primary cause.

A higher frequency of cardiac disease in the subset of stroke patients complicates ST segment alterations, which occur in [32, 33,] and 25% of individuals with ischemic stroke. [34,35]

Even without electrolyte disturbance or prior ischemic cardiac events, new T wave abnormalities are seen in 15% of acute stroke patients. [34] Up to 55% of stroke patients with the lowest prevalence of concurrent cardiac diseases have shown inverted or flat T waves. [36,37]

An autopsy investigation of 5 patients with stroke with ECG abnormalities provides more concrete evidence. None of the individuals who underwent autopsies showed signs of epicardial coronary disease. [38]

Twelve patients with sudden strokes and ST elevation in the ECG underwent thorough cardiac evaluations by Kono and colleagues. Although the echocardiography revealed anomalies in the wall motion of the individuals, there was no of coronary artery disease. [39] They also provide credence to the idea that stroke-induced ECGs are momentary and disappear over a few days to several months with no adverse affects.

Q WAVES AND U WAVES

13 - 15 % of individuals with acute ischemic stroke experience new U waves alone or in combination with T waves and QT abnormalities. [25, 41] Both ischemic and hemorrhagic strokes had isolated U waves, whereas ischemic strokes were more likely to have both U waves and QT prolongation. [44]There is no correlation between the presence of U waves and the mortality from stroke, indicating that this ECG abnormalitydidn't need to be treatment

CARDIAC INJURY AND DYSFUNCTION

Following an ischemic stroke, there was a convincing increase in cardiac tissue catecholamines, as demonstrated by Offerhaus and Van Gool. [46] These catecholamine-induced subendocardial lesions were also characterized by myofibrillar degeneration and dispersed foci of enlarged myocytes surrounded by invading monocytes.

The distinctive pathological alterations have been collectively referred to as myofibrillar degeneration, coagulative myocytolysis, or contraction band necrosis.

Myocardial necrosis usually develops as a vascular distribution in CAD patients. [47] Within minutes of the injury's commencement, neurogenic myocardial injury can be seen, and cellular abnormalities can be seen. Mononuclear infiltration, early calcification, and hypercontracted, banded cardiac cells are the most common features of myocytolysis. [47]

[48]Similar to that, diastolic dysfunction is typical after a major ischemic stroke, is related to the amount of neurological damage, and maybe the reason for the pulmonary edema these patients experience.

[49] LV dysfunction occurs early in major ischemic stroke. [50,51] After the ischemia incident, the prevalence started to diminish on days 3 to 8. According to the authors of the same study, the majority of patients' left ventricular dysfunction was completely or partially resolved during acute hospitalization. In most cases, cardiac dysfunction appears to be reversible and gradually returns to normal. [40,41].

Regional wall motion abnormality has a well-established, distinct, apical-sparing pattern that distinguishes ischemia patients from those with the typical CAD patterns.

2007 WHO World Health Report notes[52]

Worldwide, 15 million people experience a stroke every year. A third of them pass away, and a further third suffer disabilities. In the world, uncontrolled hypertension is a factor in about 12.7 million strokes. Stroke incidence is decreasing in developed nations, primarily because of reduce blood pressure. However, because of population aging, the total rate of stroke continues to be high. The most severe vascular disease complication in India is stroke, leading to severe long-term impairment and extremely devastating emotional and medical expenditures.

Global Stroke Morbidity and Mortality

The WHO estimates that 150000000 people worldwide get a stroke yearly. Five million die soon, and another five million become permanently disabled. Around the world, more than 12.7 million strokes are caused by high blood pressure.. However, because of population aging, the total rate of stroke continues to be high.

Source: World Health Organization's World Health Report for 2002. Every year, 15 million new acute strokes occur.

Stroke Morbidity and Mortality in India

In India, the prevalence of stroke is 62 per 100,000 people of all age group. Each year, there are 1.44–1.64 million new acute stroke cases, totaling 6,398,000 DALYs.

Prevalence/incidence of stroke in India

The prevalence of strokes is limited, frequently biassed, and inconsistent due to inadequate diagnostic standards. According to the area, India's crude prevalence rate varies. Urban areas have a greater rate than rural ones.

The prevalence of stroke in India 84262/100,000 in rural to 334424/100,000 in urban areas.

Overall burden of stroke in India

India continues to experience an epidemic of the stroke while the prevalence of stroke declines in emerging nations. As the incidence rises, India will have cost-related issues associated with stroke.

According to statistics, stroke accounts for between 9.5% and 30% of all neurological cases and 0.3% to 9.4% of all medical hospitalizations. According to the results of recent studies, 4% of all stroke cases occur in people under the age of 40. If they have previously experienced a stroke, older people are more susceptible to another one. Annual calculations of the cost of stroke treatment have not been exact.

MATERIALS AND METHODS

STUDY DESIGN:

Hospital-based cross-sectional descriptive study.

SOURCE OF DATA:

- The study will include Inpatients of BLDE'U Shri B .M.Patil Medical College hospital and research centre, Vijayapura

- The patients will be informed about the study in all respects, and informed consent will be obtained.

- Period of study will be from January 2021 to June 2022.

ANNEXURE-V

METHOD OF COLLECTION OF DATA

Study patients:

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- Diagnosis of cerebrovascular accident (acute ischemic stroke) which are proved by CT / MRI will be included in the study.

- Detailed history, comprehensive neurological and systemic examination will be carried out, and necessary biochemical investigation will be done

- A 12 lead ECG will be taken within 24 hours of admission.

- Results will be observed and statistical analysis will be done.

METHODOLOGY

1

- Patients with acute ischemic stroke will be studied (age>18, both genders will be involve)
- Patients with typical signs and symptoms of Acute ischemic stroke will undergo a standardized assessment with clinical history, comprehensive neurological examination and systemic examinations

2

- 12 lead ECG will be done, other laboratory investigation mentioned in proforma , CT/MRI within 24 hours , screening

ECHO will be done to all the patients with ECG changes

3

- ECG interpretation with rate , rhythm, P wave , ST segment , QRS complex , T wave amplitude and morphology

SAMPLE SIZE: 60 patients

With anticipated ECG changes observed 35% T wave inversion[1] in acute ischemic stroke. The study requires a sample size of 60 patientswith 95**%** level of confidence and 12% absolute precision**.**

Formula used

- $n=z^2 \frac{p*q}{d^2}$

Where Z= Z statistic at α level of significance d^2=

Absolute error

P= Proportion rate

q= 100-p

STATISTICAL ANALYSIS:

- The information obtained will be entered in a Microsoft Excel sheet, and statistical analysis will be Performed using a statistical package for the social sciences (Version 20).
- Results will be presented as Mean (Median) ±SD, counts and percentages and diagrams.

INCLUSION CRITERIA:

1. All the patients admitted in the medicine ward aged above 18 years irrespective of sex, because of acute ischemic stroke confirmed by clinical findings and CT&MRI revealed within 24hrs of onset of symptoms.

EXCLUSION CRITERIA:

1. Old stroke
2. Ischemic heart disease
3. Hemorrhagic stroke
4. Recurrent stroke
5. Electrolyte imbalance
6. K/C/O Hypertension

RESULTS

Table 1: AGE WISE DISTRIBUTION OF STUDY

Age(Years)	No. of patients	Percentage
<= 30	2	3.3
31 - 40	5	8.3
41 - 50	6	10.0
51 - 60	14	23.3
61 - 70	19	31.7
71+	14	23.3
Total	60	100.0

Total study subjects participated in the study were 60 , the highest percentage of study subjects were in the age group of 61– 70 years .The mean and standard deviation is 60 and 13.8 respectively

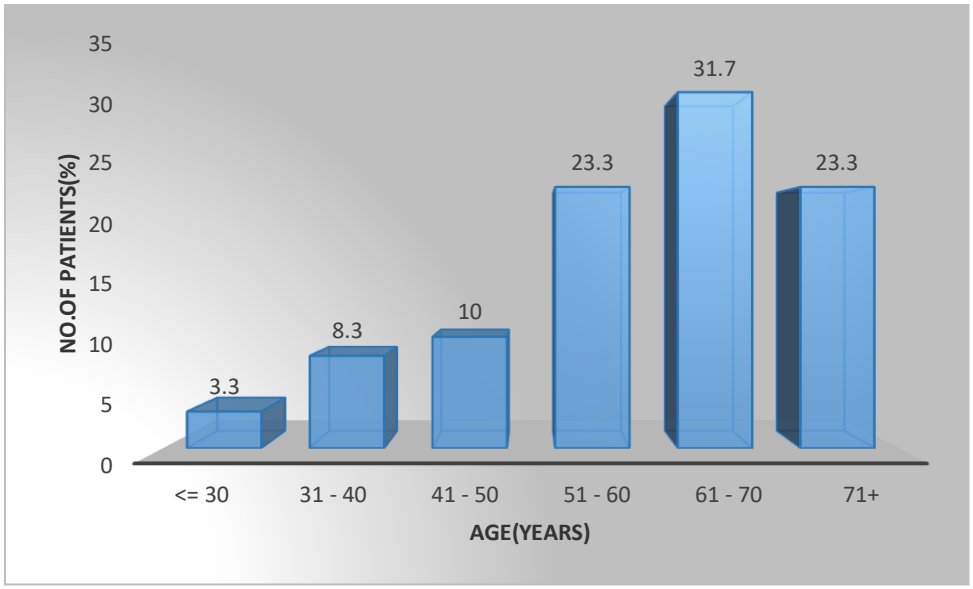


Table 2:GENDER WISE DISTRIBUTION OF STUDY

Gender	No9of patients	Percentage
Female	23	38.3
Male	37	61.7
Total	60	100.0

The highest percentage of study subjects were Male with 61.7%

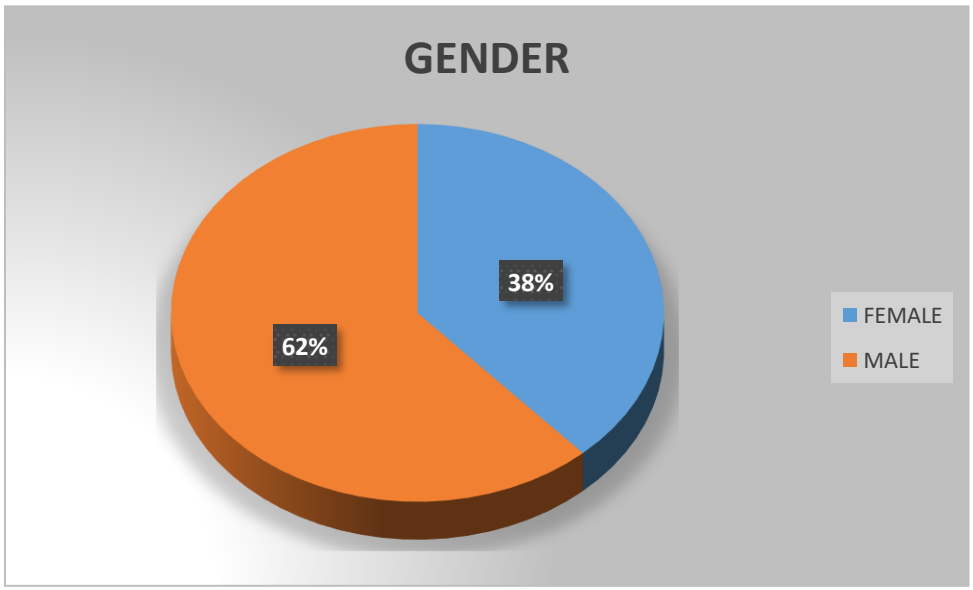


Table 3: LOCATION AND TYPE OF CEREBROVASCULAR LESION WITH NO OF PATIENTS AND PERCENTAGE

DIAGNOSIS	No. of patients	Percentage
BASAL GANGLIA INFARCT	2	3.3
THALAMUS,CAPSULOGANGLIONIC INFARCT	1	1.7
CAPSULO GANGLIONIC INFARCT	12	20.0
CAUDATE NUCLEUS INFARCT	1	1.7
CENTRUM,SEMIOVALE INFARCT	1	1.7
CEREBELLAR INFARCT	2	3.3
CORONA RADIATA INFARCT	3	5.0
FRONTAL LOBE INFARCT	2	3.3
FRONTO PARIETO INFARCT	3	5.0
FRONTO-TEMPORAL INFARCT	11	18.3
FRONTO-TEMPORO-PARIETAL INFARCT	1	1.7
LENTIFORM NUCLEUS INFARCT	1	1.7
INSULAR CORTEX WITH PUTAMEN INFARCT	1	1.7
LATERAL MEDULLARY SYNDROME	1	1.7
LENTIFORM NUCLEUS,INTERNAL CAPSULE INFARCT	1	1.7
OCCIPITAL ,MIDBRAIN,PONS INFARCT	1	1.7
PARIETAL LOBE INFARCT	1	1.7
PARIETO OCCIPITAL LOBE INFARCT	8	13.3
PARIETO-TEMPORAL INFARCT	5	8.3

TEMPORAL LOBE INFARCT	1	1.7
THALAMUS AND TEMPORO-PARIETAL INFARCT	1	1.7
Total	60	100.0

The highest percentage of patients had capsulo ganglionic infarct ,20% , followed by fronto – temporal infarct , 18.3%

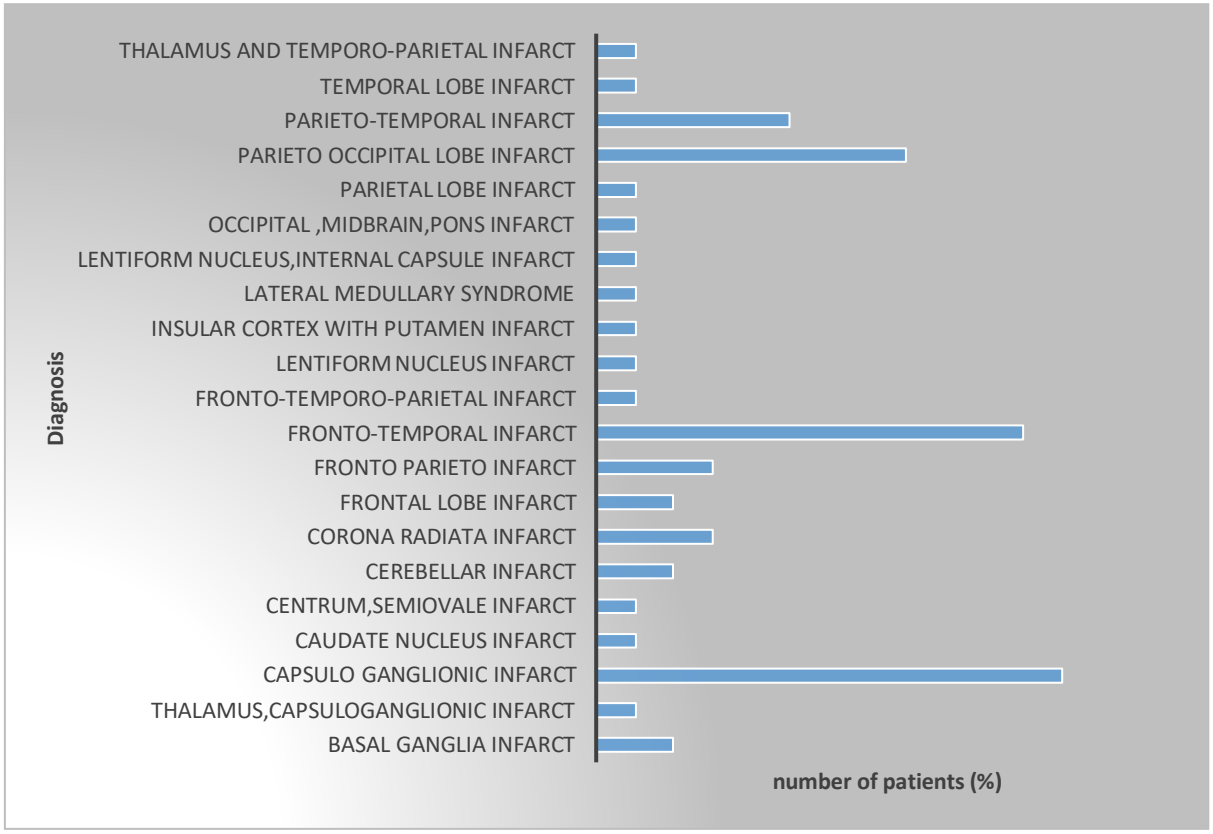


Table 4: THE INCIDENCE OF RHYTHM CHANGES IN THE STUDY GROUP

Rhythm	No of patients	Percentage
IRREGULAR	7	11.7
Normal	53	88.3
Total	60	100.0

Rhythm disturbance were present in 11.7% of patients

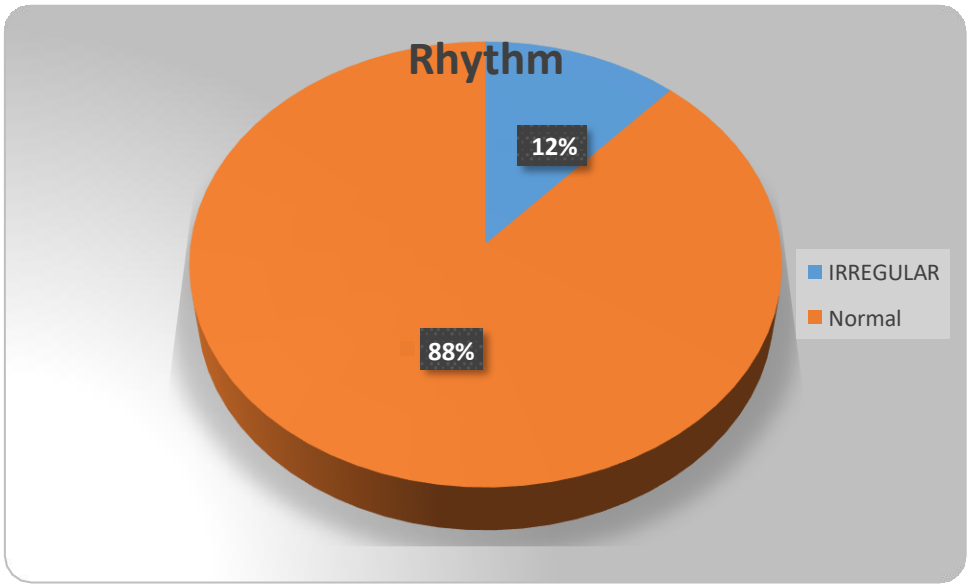


Table 5: THE INCIDENCE OF P WAVE CHANGES IN THE STUDY GROUP

P wave	No. of patients	Percentage
0.12	1	1.7
Normal	59	98.3
Total	60	100.0

P wave changes were present in 1.7% 0f patients.

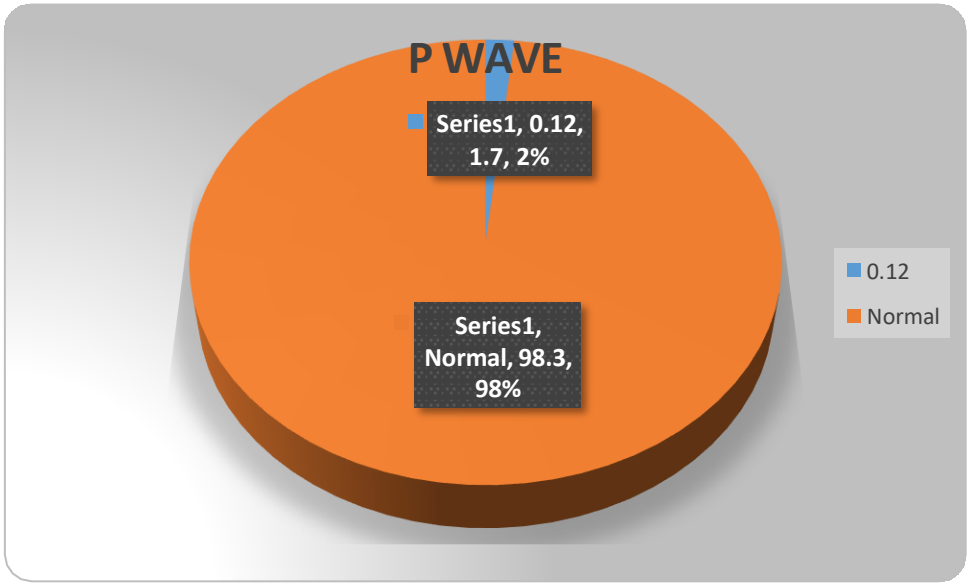


Table 6 :THE INCIDENCE OF PR INTERVAL CHANGES IN THE STUDY GROUP

PR interval	No. of patients	Percentage
Normal	60	100.0

None of the patients in our study group have abnormal PR interval

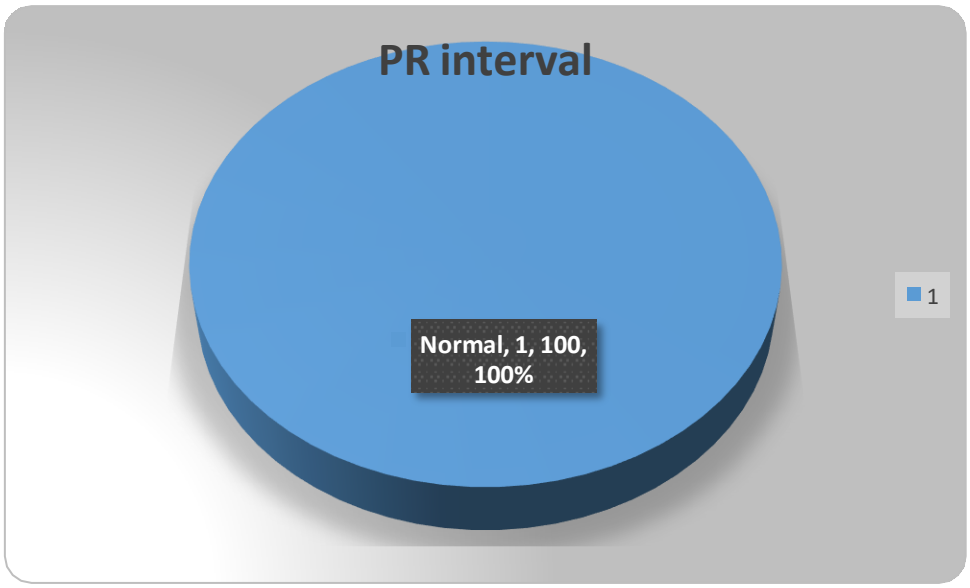


Table 7: THE INCIDENCE OF QRS CHANGES IN THE STUDY GROUP

QRS Complex	No. of patients	Percentage
Normal	60	100.0

None of the patients in our study group have abnormal QRS complexs

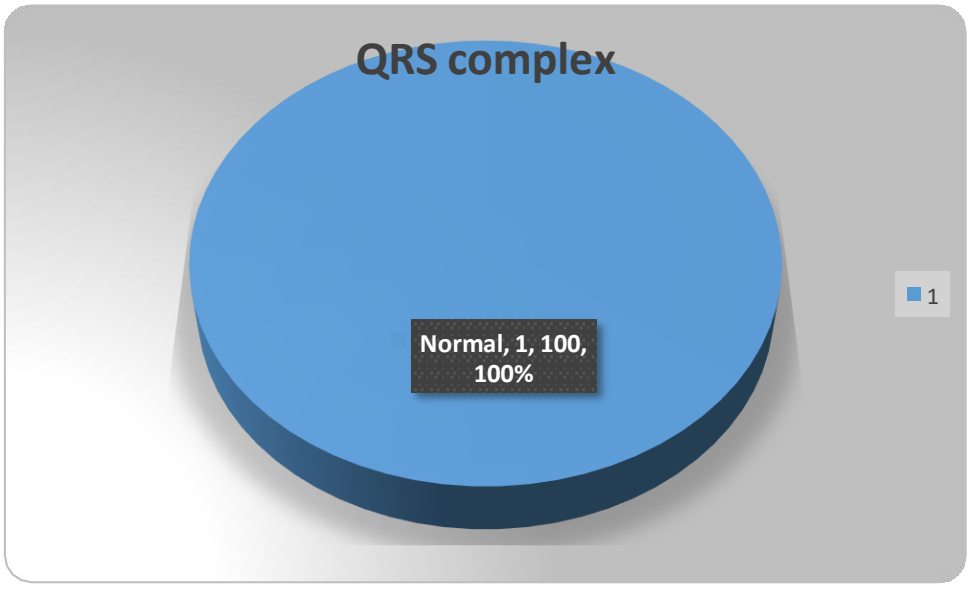


Table 8: : THE INCIDENCE OF ST SEGMENT CHANGES IN THE STUDY GROUP

ST segment	No. of patients	Percentage
DEPRESSED 1,AVL,V5,V6	1	1.7
DEPRESSED I,AVL	4	6.7
DEPRESSED II,III,AVF	3	5
DEPRESSED V2,V4	2	3.3
DEPRESSED V4-V6	1	1.7
ELEVATED V2-V4	2	3.3
ELEVATED V5-V6	3	5.0
Normal	44	73.3
Total	60	100.0

6.7 % patients have depression of ST segment in I ,aVL

5 % patients have elevated of ST segment in V5-V6

5% patients have depression of ST segment in II,III,Avf

3.3% patients have depression of ST segment in V2-V4

3.3% patients have elevation of ST segment in V2-V4

1.7% patients have depression of ST segment in I ,aVL, V4-V6

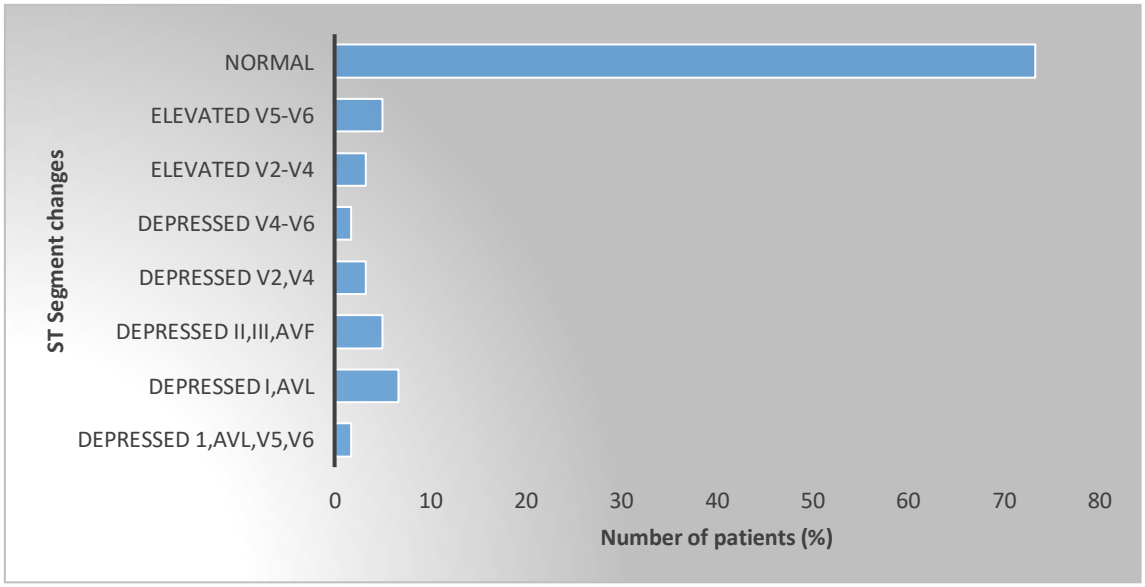


Table 9: : THE INCIDENCE OF T WAVE CHANGES IN THE STUDY GROUP

T wave	No. of patients	Percentage
TALL V2,V3,V4	1	1.7
INVERTED I,AVL	4	6.7
INVERTED I,AVL,V5,V6	3	5.0
INVERTED II,III,AVF	5	8.3
INVERTED V1-V4	1	1.7
INVERTED V2-V6	2	3.3
INVERTED V4-V6	2	3.3
Normal	42	70.0
Total	60	100.0

8.3% patients have inversion of T wave inII,III,AVF

6.7% patients have inversion of T wave in I,AvL

5% patients have inversion of T wave in I,AvL , V5-V6

3.3% patients have inversion of T wave in V2-V6 , V4-V6

1.7% patients have inversion of T wave inV1-V4

1.7% patients haveTall T wave inV2-V4

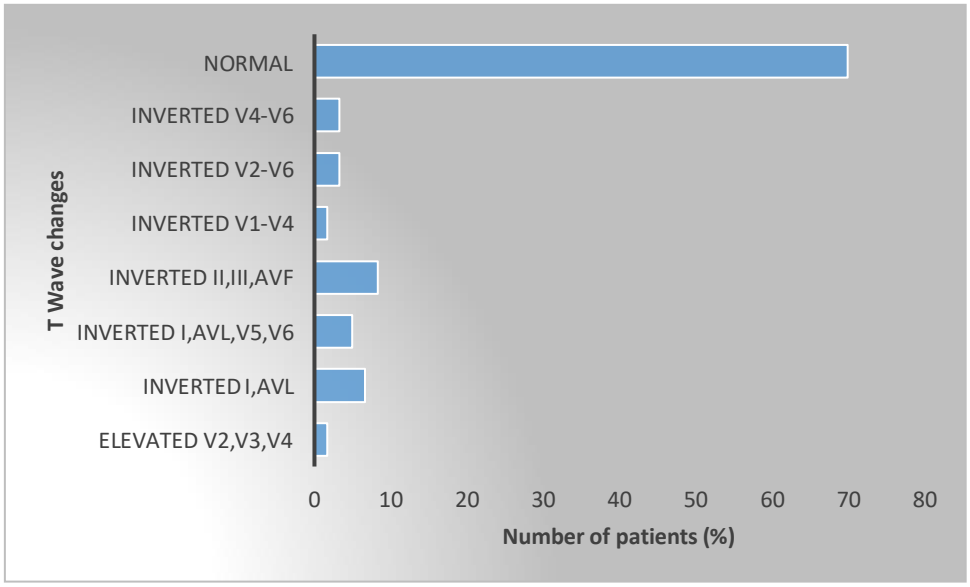


Table 10: : THE INCIDENCE OF RATE CHANGES IN THE STUDY GROUP

RATE	No. of patients	Percentage
<= 60	16	26.7
61 - 100	36	60.0
101+	8	13.3
Total	60	100.0

26.7% of the study group have bradycardia

13% of the study group have tachycardia

60% of the study group were normal

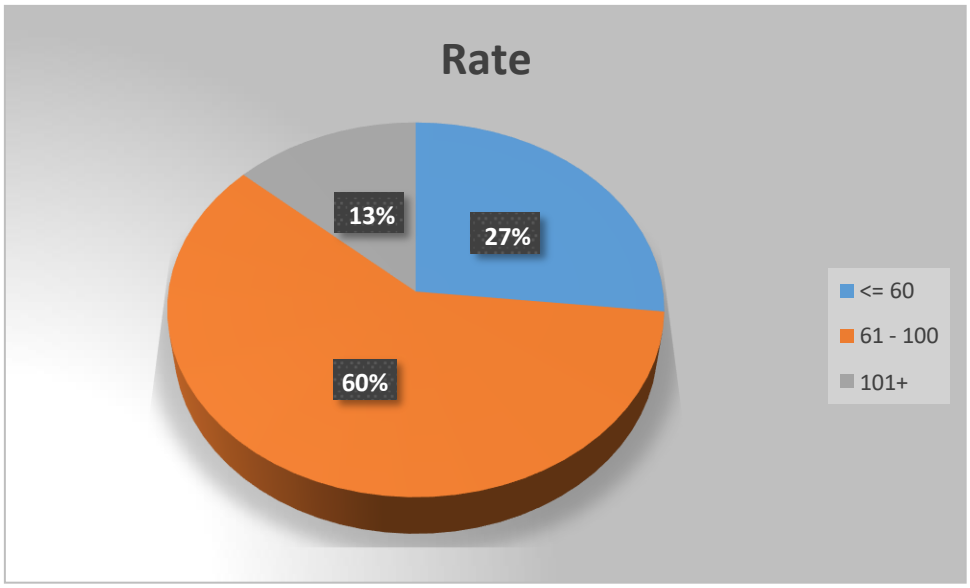


Table 11: : THE INCIDENCE OF QTc CHANGES IN THE STUDY GROUP

QTc interval	No. of patients	Percentage
<0.36	2	3.3
0.36 - 0.45	29	48.3
0.46+	29	48.3
Total	60	100.0

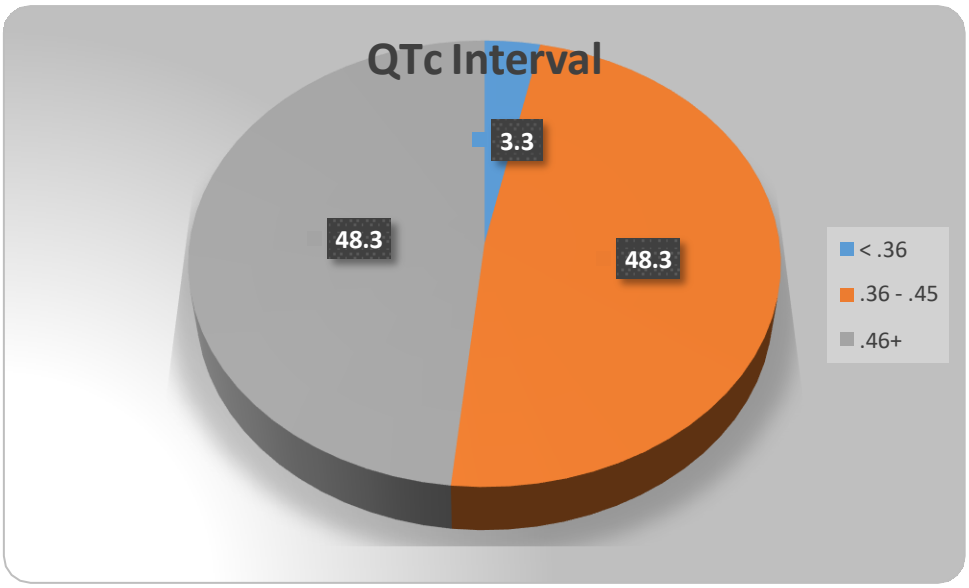
3.3% in the study group have narrow QTc interval

48.3%in the study group have prolonged QTc interval

48.3%in the study group have normal QTc interval

Mean in the study group is 0.47

Standard deviation in study group 0.091



Descriptives

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
AGE	60	22	85	60.52	13.869
RATE	60	46	120	76.93	20.468
QTc INTERVAL	60	.3400	.6680	.475783	.0915586
Valid N (listwise)	60				

DISCUSSION

The present study group , the mean age was 60.52 years

Kumar et al[53] reported a mean age of 63±12.68 years in their study, while Saxena et al[54] reported a mean age of 67±8.90 years.

In the study group 21.6% were in patients less than 50 years of age (young strokes)

ECG was normal in 11.25% cases. Goldstein et al[55] reported 8% of cases of CVA had no ECG changes, Purshothaman et al[56] reported normal ECG in 22% cases while Dimant et al[17] reported that 10% of cases of CVA had no ECG changes.

In the study group , 28.3% have T wave inversions,%1.7% have Tall T wave

T wave inversions were reported in 36.1 % cases by Kumar et al[53] while Tandur and Sundragiri[57] reported T wave inversions in 24 % cases. Togha et al[58] reported T wave inversions in 39.9% cases, Purshothaman et al[56] in 34.8% cases, Somasundaram[59] in 22.3% cases .Cruickshank et al, [60] who observed Tall T waves

In the study group 18.4 % have ST depression,8.3% have ST elevation

Kumar et al[53] reported ST changes in 16.4% cases, Tandur et al[57] in 20% cases, Golstein et al[55] reported ST changes in 39% cases and Dogan et al[61] in 65% cases. Mc Dermott et al[62], reported ST changes inonly 8% of cases of CVA .Frentz and Gorsmen 15% with infarction and also studyof Lindgren Et Al who showed ST segment depression in lateralleads.

In the study group 48.3% have QTc prolongation,3.3 % have narrow QTc interval

Togha et al[58] reported QT prolongation in 32.4% cases . Golstein et al[55] detected QT prolongation in 45% cases while Dogan et al[61] reported QT prolongation in 26% cases.Arruda and Lacerda[63] which showed 67% of patients with ischemic stroke and also study of Keller and Williams [64]in patients with stroke.

In the study group none of the patients have abnormal QRS complexs

Purshothaman et al[56] reported normal QRS complex 12% cases , Toga M et al reported normal QRS complex 10.5% cases

In the study group none of the patients have abnormal PR interval and P wave

Sundragiri[16] reported normal PR interval and P wave in 24 % cases

In the study group Rhythm disturbance was observed in 11.7% . This is in correlation with study of Chou et al[64] and Crop and Manning et al .

In the study group 26.7 % have Sinus bradycardia. This is observed in study of stober and associates described sinus bradycardiain 23% of patients.

In the study group no specific correlation of ECG changes with site of cerebral lesion .this study is observed in Rambabu et al

All these patients with ECG abnormalities, a screening echo were performed to rule out cardiac abnormalities associated.

These results imply that the central nervous system's structures involved in cardiovascular function are extensively dispersed. Therefore, it is possible that CVA lesions can damage or irritate such widely dispersed neurons or pathways regulating the cardiovascular system, resulting in changes to the ECG, not only in the frontal lobe but also in the temporo-parietal lobe and basal ganglia.

CONCLUSION

- The following conclusions can be drawn from the results of this study
- QTc prolongation changes , 48.3% were the most common abnormality noted in the study.
- Next common abnormality noted was T wave inversion which was noted in 28.3%% of patient.
- Sinus Bradycardia were noted in 26.7% of patients.
- ST depression were noted in 18.4% of patients.
- Sinus tachycardia were noted in 13.3% of patients
- None of the patients in our study had associated electrolyte abnormalities.
- These ECG changes were not associated with any particular site cerebral lesion.
- Different types of cardiac disturbance are common when there has been an acute neurological injury. Following an ischemic stroke, there was increase in cardiac tissue catecholamines. These catecholamine-induced subendocardial lesions were also characterized by myofibrillar degeneration and dispersed foci of enlarged myocytes surrounded by invading monocytes.

- The distinctive pathological alterations have been collectively referred to as myofibrillar degeneration, coagulative myocytolysis, or contraction band necrosis.
- [47] Within minutes of the injury's commencement, neurogenic myocardial injury can be seen, and cellular abnormalities can be seen. Mononuclear infiltration, early calcification, and hypercontracted, banded cardiac cells are the most common features of myocytolysis. [47]
- More importantly, the existence ofcardiac abnormalities has a significant effect on clinical management and affects the result of the cardiac and nervous system.

- The study confirms the role of ECG in workup of acute ischemic stroke patients. Electrocardiographic changes are very common in cases of acute ischemic stroke, even in those having no history of coronary heart disease ,understanding that these ECG which are occurring in patients with CVA is important because it may lead to erroneous judgment of assigning these patients as CAD. Interpreting these ECG changes can aid the treatment of patientswithrespect of revascularization and surgical interventions.

- Though our study is by no means exhaustive, it does provide a glimpse into the variety of ECG changes in the absence of any cardiac disease in acute ischemic stroke. Although we understand to some extent these changes and also since few studies have been done on this aspect, further research is needed to study the pattern of ECG changes in acute ischemic stroke.

SUMMARY

The study entitled " Study the pattern of Electrocardiographic changes in Acute Ischemic Stroke " was conducted in the department of medicine, BLDE(Deemed to be University) Shri B.M Patil medical college, hospital and research centre, vijayapura during the period from 2021-2022. The aim of the study was to study the pattern of electrocardiographic changes in Acute Ischemic Stroke.

The study included 60 patienys, age of more than 18 years, selected from inpatient department from Shri B.M. Patil medical college, hospital and research centre, Vijayapura.

After taking consent and a detailed history from the subjects,12 lead electrocardiogram,CT/MRI brain within 24 hours ,screening ECHO will be done to all the patients with ECG changes . The ECG results were evaluated for different parameters like heart rate, P wave, PR interval, QRS complex, QT interval, QTc interval, ST segment, Q wave and T wave.

The results were as follows:

QTc prolongation changes , 48.3% were the most common abnormality notedin the study.Next common abnormality noted was T wave inversion which was noted in 28.3%% of patient.Sinus Bradycardia were noted in 26.7% of patients. ST depression were noted in 18.4% ofpatients.Sinus tachycardia were noted in 13.3% of patients None of the patients in our study had associated electrolyte abnormalities.These ECG changeswere not related with particular siteof cerebral lesion.Though our study is by no means exhaustive, it does provide a glimpse into the variety of ECG changes in the absence of any cardiac disease in acute ischemic stroke. Although we understandto some extent these changes and also sincefew studies have been done on this aspect, further research is needed to study the pattern of ECG changes in acute ischemic stroke .

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. **ANNEXURE – I**

ETHICAL CLEARANCE CERTIFICATE

B.L.D.E. (DEEMED TO BE UNIVERSITY) IEC/NO-09/2021 Date- 22/01/2021

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11am to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: Study of Electrocardiographic changes in acute ischemic stroke

Name of PG student: Dr Mayuri M Bhujappagol, Department of Medicine

Name of Guide/Co-investigator: Dr V G Warad , Professor of Medicine

DR S.V.PATIL
CHAIRMAN
Institutional Ethical Committee
B L D E (Deemed to be University)
Shri B.M. Patil Medical College,
VIJAYAPUR-586103 (Karnataka)

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

ANNEXURE – II

INFORMED CONSENT FORM

TITLE OF RESEARCH: STUDY OF ELECTRCARDIOGRAPHIC CHANGES IN ACUTE ISCHEMIC STROKE

GUIDE : DR VIJAYKUMAR G WARAD,M.D

P.G.STUDENT : DR MAYURI MALAGOUDA BHUJAPPAGOL

All aspects of this consent form are explained to the patient in the language understood by him or her.

PURPOSE OF STUDY:

I have been informed that the purpose of this study is to study the pattern of Electrocardiographic changes in Acute Ischemic Stroke.

.

PROCEDURE:

I understand that I will undergo detailed history and clinical examination and investigations

BENEFITS:

I understand that my participation in this study will have no direct benefit tome other than the potential benefit of treatment which is planned to prevent further morbidity and mortality in me.

CONFIDENTIALITY:

I understand that the medical information produced by the study will become a part of hospital record and will be subjected to confidentiality and privacy regulation of hospital. If the data is used for publication the identity will not be revealed.

REQUEST FOR MORE INFORMATION:

I understand that I may ask for more information about the study at any time.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or withdraw from study at any time.

(Signature of patient) (Signature of Guardian)

STUDY SUBJECT CONSENT FORM:

I confirm that **Dr.MAYURI MALAGOUDA BHUJAPPAGOL** has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all above in detail in my own language and I understand the same. I agree to give my consent to participate as a subject in this research project.

DATE: 30.11.2022 DATE:30.11.2022

SIGNATURE OF PARTICIPANT : SIGNATURE OF WITNESS:

ANNEXURE – III

PROFORMA

SCHEME OF CASE TAKING

Name: CASE NO:

Age: OP/IP NO:

Sex: DOA:

Religion: DOD:

Occupation:

Address:

Presenting complaints with duration:

History of presenting complaints:

Past History:

Family History:

Personal History:

Treatment History:

General Physical Examination

Pallor: present/absent
Icterus: present/absent
Cyanosis: present/absent
Clubbing: present/absent
Generalized lymphadenopathy: present/absent
Odema: present/absent

VITALS:

PR:

BP: in mm of mercury (mm hg) RR:

Temp:

SYSTEMIC EXAMINATION:

- Cardiovascular system
- Respiratory system
- Per abdomen
- Central nervous system

INVESTIGATIONS PATHOLOGY:

1.)Complete blood count:	
Hb	gm/dl
Total count	Cells/cumm
Differential count	
Neutrophils	%
Lymphocytes	%
Eosinophils	%
Basophils	%
Monocytes	%
2.) ESR	At the end of 1st hour.
3.)Platelet Indices	
Platelet Count	
Mean Platelet Volume	
Platelet Distribution Width	
4.) Urine Routine	
Sugar	
Albumin	
Cell type	
Cell count	

BIOCHEMISTRY:

RBS	
Blood urea	
Serum creatinine	
Serum sodium	

Serum potassium	
Serum chloride	
serum uric acid	

TROPONIN I AND CPK-MB

2D ECHO

ELECTROCARDIOGRAPHY:

CHEST X RAY PA VIEW

Other relevant investigations will be done when required.

CONCLUSION:

DATE: 30.11.2022 **SIGNATURE**

ANNEXURE – IV : MASTERCHART

SL NO	NAME	AGE	SEX	IP NUMBE	DIAGNOSIS	RATE	RHYTHM	P WAV	PR INTERV	QRS COMP	ST SEGMENT	T WAVE	QTc INTERVAL
1	HUSEN BADAF	70	M	20712	LEFT CAPSULO GANGLIONIC INFARCT	54	N	N	N	N	N	INVERTED I,AVL,V5,V	0.36
2	DUNDAWWA	80	F	19807	RIGHT INFARCT IN LENTIFORM NUCLEUS	110	N	N	N	N	DEPRESSED I,AVL,V5,	N	0.4
3	LAXMIBAI	58	F	24229	RIGHT LATERAL MEDULLARY SYNDROME	78	N	N	N	N	N	INVERTED II,III,AVF	0.43
4	ANNAPPA	70	M	142084	LEFT PARIETAL LOBE INFARCT	86	N	N	N	N	N	N	0.52
5	CHANDRASHEKHAR	65	M	135367	LEFT PARIETO-TEMPORAL INFARCT	120	N	N	N	N	N	N	0.38
6	SHIVALINGAYYA	65	M	127365	BILATERAL CAPSULO-GANGLIONIC INFARCT	53	N	N	N	N	N	ELEVATED V2,V3,V4	0.46
7	SHARNAPPA	65	M	26124	RIGHT FRONTO-PARIETO-OCCIPITAL INFARCT	56	N	N	N	N	ELEVATED V5-V6	N	0.35
8	RAMESH BADIGER	40	M	37102	LEFT LENTIFORM NUCLEUS,INTERNAL CAPSULE INFA	90	N	N	N	N	N	N	0.44
9	DRAKSHAYANI GANACHAV	67	F	156102	LEFT PARIETO OCCIPITAL INFARCT	112	N	N	N	N	DEPRESSED I,AVL	N	0.39
10	SHRIMANTH	70	M	15608	BILATERAL FRONTO-TEMPORAL INFARCT	76	N	N	N	N	N	N	0.34
11	SHIVANAND TAPAPUR	55	M	16501	RIGHT PARIETOTEMPORAL INFARCT	80	IRREGULAI	N	N	N	N	N	0.5
12	SHAVANTRAWA PATIL	65	F	16507	BILATERAL CORONA RADIATA INFARCT	50	N	0.12	N	N	ELEVATED IN V2,V3,V4	N	0.48
13	JAIBANDER	55	F	24625	LEFT THALAMUS AND TEMPORO-PARIETAL INFARCT	84	N	N	N	N	N	INVERTED V3-V6	0.6
14	BASAMMA	73	F	22481	LEFT PARIETO TEMPORAL INFARCT	76	N	N	N	N	N	N	0.66
15	SHIVAYOGI HATTI	70	M	20075	LEFT CAUDATE NUCLEUS INFARCT	54	N	N	N	N	DEPRESSED V4-V6	N	0.46
16	RAMU RATHOD	55	M	25337	RIGHT CAUDATE NUCLEUS,CAPSULOGANGLIONIC INF	106	N	N	N	N	N	N	0.65
17	SHANTAPPA LAXMAN	70	M	22336	LEFT FRONTOPARIETAL INFARCT	114	IRREGULAI	N	N	N	N	N	0.45
18	KASHIBAI	61	F	149242	LEFT CAPSULO GANGLIONIC INFARCT	75	N	N	N	N	N	N	0.396
19	SHARADABAI MALLAPA	26	F	25477	LEFT FRONTOPARIETAL INFARCT	86	N	N	N	N	N	N	0.412
20	APPASAHEB KUCHANUR	59	M	25346	LEFT THALAMS,CAPSULOGANGLIONIC INFARCT	79	N	N	N	N	N	DEPRESSED I,AVL	0.365
21	MOHAN TILGUL	76	M	24718	RIGHT CENTRUM,SEMIOVALE INFARCT	80	IRREGULAI	N	N	N	DEPRESSED I,AVL	N	0.402
22	BASAVARAJ KATTIMANI	40	M	24460	RIGHT BASAL GANGLIA,INTERNAL CAPSULE,THALAM	90	N	N	N	N	N	N	0.396
23	HANAMANTH MARADI	46	M	160653	LEFT FRONTOPARIETAL INFARCT	78	N	N	N	N	DEPRESSED V5-V6	INVERTED I,AVL	0.402
24	PREMASINGH KHATEWADI	85	F	185000	RIGHT PARIETO-OCCIPITAL , THALAMUS INFARCT	77	N	N	N	N	N	N	0.56
25	LACHAPPA	44	M	184979	RIGHT PARIETO OCCIPITAL,THALAMUS INFARCT	88	N	N	N	N	N	N	0.662
26	VIDYA BYAKOD	32	F	277127	BILATERAL THALAMUS,CAPSULOGANGLIONIC INFARI	84	N	N	N	N	N	INVERTED II,III,AVF	0.452
27	SHEKAPPA HUKKERI	55	M	250651	LEFT PARIETAL LOBE INFARCT	70	N	N	N	N	N	N	0.398
28	MANJULA TABBENAVAR	55	F	22064	RIGHT CAPSULAR INFARCT	90	N	N	N	N	DEPRESSED I,AVL	INVERTED V4-V6	0.56
29	MAMTAZ KHAN	50	F	42161	RIGHT TEMPORO PARIETAL INFARCT	91	N	N	N	N	DEPRESSED II,III,AVF	N	0.36
30	SHIVASHANKAR	62	M	84990	RIGHT CAPSULO-GANGLIONIC INFARCT	78	N	N	N	N	N	N	0.412
31	BAPU MANE	56	M	94865	LEFT BASAL GANGLIA INFARCT	54	N	N	N	N	N	N	0.544
32	ASHOK PATTANSHETTI	55	M	101728	LEFT CAPSULO GANGLIONIC INFARCT	62	N	N	N	N	N	INVERTED IN II,III,AVF	0.568
33	SHRISHAIL	65	M	20090	LEFT CEREBELLAR INFARCT	94	IRREGULAI	N	N	N	DEPRESSED V5,V6	N	0.402
34	GANGARAM	68	M	94283	RIGHT CORONA RADIATA INFARCT	54	N	N	N	N	N	INVERTED I,AVL,V5,V	0.448
35	SARUBAI	68	F	16707	LEFT CAPSULO GANGLIONIC INFARCT	46	N	N	N	N	N	N	0.543
36	SHRANAPPA	70	M	162742	RIGHT PARIETO TEMPORAL INFARCT	120	N	N	N	N	N	N	0.42
37	KASHISAB	62	M	165783	RIGHT OCCIPITO TEMPORAL INFARCT	89	N	N	N	N	N	INVERTED IN I,AVL	0.55
38	VISHWARAPPA	72	M	136868	RIGHT FRONTO PARIETAL INFARCT	46	N	N	N	N	N	N	0.396
39	TOOLAMA	45	F	128772	RIGHT INSULAR CORTEX WITH PUTAMEN INFARCT	85	N	N	N	N	N	INVERTED I,AVL,V2-V	0.47
40	MANOHAR	76	M	10086	RIGHT FRONTAL LOBE INFARCT	76	N	N	N	N	DEPRESSED II,III,AVF	N	0.56
41	SHANKARGOUDA PATIL	40	M	97216	LEFT CAPSULO GANGLIONIC INFARCT	114	N	N	N	N	N	INVERTED V1-V4	0.62
42	NOOR JAAN	53	F	72684	RIGHT CAPSULO-GANGLIONIC INFARCT	77	N	I	N	N	N	N	0.665
43	SAROJANEBAI	76	F	46360	LEFT PARIETAL LOBE INFARCT	54	N	N	N	N	N	INVERTED II,III,AVF	0.396
44	LAGMAVVA	75	M	112386	LEFT CAPSULO GANGLIONIC INFARCT	96	IRREGULAI	N	N	N	N	N	0.449
45	ASHOK	60	M	11876	RIGHT FRONTO TEPMPORO PARIETAL INFARCT	46	N	N	N	N	N	INVERTED V2-V6	0.424
46	LATABAI	74	F	12610	B/L CORONA RADIATA INFARCT	74	N	N	N	N	DEPRESSED V2,V4	N	0.398
47	SAVITRI	72	F	112319	RIGHT FRONTAL LOBE INFARCT	63	N	N	N	N	DEPRESSED I,AVL	N	0.491
48	SHANTA	48	F	113555	LRFT PARIETAL LOBE INFARCT	76	N	N	N	N	N	N	0.502
49	SIDDAVA	65	F	122366	RIGHT FRONTO TEPMPORO PARIETAL INFARCT	116	N	N	N	N	N	INVERTED II,III,AVF	0.512
50	ARAVIND	75	M	73870	RIGHT FRONTOPARIETAL INFARCT	68	N	N	N	N	DEPRESSED V2-V4	N	0.467
51	BAGAPPA	60	M	33277	RIGHT FRONTO TEMPORAL INFARCT	60	N	N	N	N	N	N	0.394
52	BASALINGAPPA	75	M	114563	LEFT FRONTAL ,GENU OF CORPUS CALLOSUM	78	N	N	N	N	N	N	0.39
53	BASAMMA KUMBAR	80	F	68037	LEFT FRONTAL LOBE INFARCT	94	IRREGULAI	N	N	N	N	N	0.403
54	BERAPPA PUJARI	52	M	56903	RIGHT TEMPORAL LOBE INFARCT	46	N	N	N	N	N	N	0.62
55	HANAMANTH MADALLI	22	M	72895	LEFT FRONTO-TRMPORO-PARIETAL-OCCIPITAL INFA	70	N	N	N	N	DEPRESSES II,III,AVF	N	0.48
56	ISMAIL	35	M	56992	LEFT FRONTAL LOBE INFARCT	56	N	N	N	N	N	INVERTED I,AVL	0.446
57	RAVI	45	M	65319	RIGHT PARIETAL ,CAPSULO GANGLIONIC INFARCT	48	N	N	N	N	ELEVATED V2-V6	N	0.5
58	SHAKUNTALA	73	F	95959	BILATERAL CEREBELLAR INFARCT	63	N	N	N	N	N	N	0.566
59	SHRISHAILAYYA	70	M	47384	RIGHT FRONTO-TEMPORO-PARIETAL INFARCT	76	N	N	N	N	N	N	0.608
60	SIDAMMA	60	F	68646	RIGHT OCCIPITAL ,MIDBRAIN,PONS INFARCT	50	IRREGULAI	N	N	N	N	INVERTED V1-V6	0.668